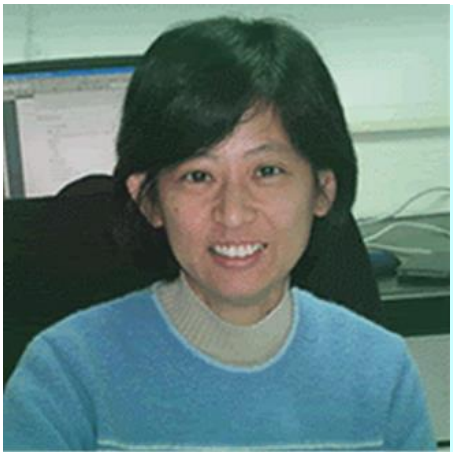

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## 研究興趣及成果簡述

胸膜肺炎放線桿菌(*Actinobacillus pleuropneumoniae*)是豬隻胸膜肺炎的病原，世界各國均有本病發生，往往造成養豬業重大經濟損失。胸膜肺炎放線桿菌產生多種毒性因子，其中外毒素Apx為主要致病因子。本菌依血清型不同，生成二至三種Apx，這些外毒素除具有不等程度之溶血性及急性細胞毒殺效果，亦傷害肺臟內皮細胞及第一線防衛細胞-肺泡巨噬細胞。相較於其他Apx毒素，ApxI之溶血性與細胞毒性均屬最高，為極重要毒性因子。本實驗室研究方向主要著重於ApxI對宿主細胞造成傷害之機制，探討ApxI對豬肺泡巨噬細胞之影響，並釐清相關訊息傳導路徑，包括毒素受體及下游訊息分子之確認，期深入瞭解ApxI在胸膜肺炎放線桿菌致病機制扮演之角色，以利本病防治策略之擬定。目前已證實低濃度ApxI可誘發豬肺泡巨噬細胞凋亡及前炎症細胞素IL-1β、IL-8、TNFα之表現；ApxI藉由MAPK (p38及JNK)活化caspases 3、8、9，繼而誘導細胞凋亡，即外源性及內源性凋亡路徑均被活化，而MAPK及NF-κB亦調控前炎症細胞素之表現。此外。近期研究發現豬源LFA-1(CD11a/CD18)可能為ApxI受體，ApxI與LFA-1具交互作用，並抑制下游FAK與Akt活性，進而導致細胞死亡，然而ApxI係以何結構區與LFA-1次單位行交互作用則仍待釐清。

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